

Clinical Management Review

Clinical Management of Atopic Dermatitis: Practical Highlights and Updates for the Atopic Dermatitis Practice Parameter 2012

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Atopic dermatitis is a challenging condition for clinicians and patients. Recent advances were documented in the Atopic Dermatitis Practice Parameter 2012, and we want to provide clinicians with key points from the Atopic Dermatitis Practice Parameter 2012. In this article, we highlight the evidence-based therapy of atopic dermatitis as well as provide practical tips for clinicians and families. An updated review of immunopathology provides a firm basis for patient education and therapy. We also review clinical diagnosis and ways to improve quality of life for patients with atopic dermatitis. © 2014 American Academy of Allergy, Asthma & Immunology (J Allergy Clin Immunol Pract 2014;■:■-■)

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Atopic dermatitis (AD) is often the first manifestation of allergic disease. Most patients with AD also will develop another atopic disorder, such as allergic rhinitis, asthma, or food allergy. The evaluation and management of AD are integral parts of an

allergist/immunologist's training and practice. In this review, we highlight the key points for the clinician from the Atopic Dermatitis Practice Parameter 2012 (ADPP).¹ The immunopathology and genetics of AD guide the treatment, and updates are briefly summarized. We review the clinical diagnosis and treatment of AD from the ADPP and highlight practical tips for the treatment recommendations.¹ The major decision points in the management and therapy of AD are shown in Figure 1 (from the ADPP).

IMMUNOPATHOLOGY AND GENETICS

AD is associated with impaired skin barrier function, a proinflammatory atopic response to antigens, and reduced cutaneous antimicrobial activity. Because these 3 main factors and their molecular pathways interact and potentiate each other, optimal AD management addresses all 3 simultaneously. Skin barrier dysfunction in AD gained attention as a primary component of the disease in 2006, when filaggrin gene (*FLG*) nonsense mutations were found in patients with AD and ichthyosis vulgaris.² Since then, abnormalities in epidermal protein function have been identified in loricrin and involucrin,³ the profilaggrin-like protein hornerin, *FLG* family member 2, and in 2 *FLG*-like proteins.⁴ All these proteins are found at reduced levels not only in lesional skin but also in nonlesional skin, and all are down-regulated in response to the Th2 cytokines known to be elevated in AD.³⁻⁵ Uninflamed AD skin has reduced lipids⁶ as well as reduced barrier function at baseline. Abnormal lipid content and protease activity can be explained at least in part by a defect in *Tmem79*, a gene that controls lamellar granule transport function in the epidermal granular layer, just below the stratum corneum.⁷ Cellular tight junctions in the granular layer also are abnormal; single nucleotide polymorphisms were found in the tight junction claudin-1 gene.⁸ Protein expression of claudin 1 and claudin 23 was decreased at baseline and further decreased in response to Th2 cytokines. However, an abnormality in the epidermal barrier alone is not sufficient for development of AD because even patients homozygous for filaggrin null mutations do not always develop dermatitis or can clinically outgrow the disease.⁹

An impaired skin barrier permits increased entry of microbes, chemical antigens, and irritants, which leads to a potent and prolonged proinflammatory Th2 cytokine environment that involves elevated levels of IgE, IL-4, IL-5, IL-13, and IL-31.¹⁰ Because these cytokines downregulate epidermal proteins, abnormalities in epidermal integrity and cytokine activity promote each other.^{3,4} It has been shown that the Th2 response is mediated by IL-25, IL-33, and thymic stromal lymphopoietin (TSLP) released by barrier-disrupted epidermis or tissue

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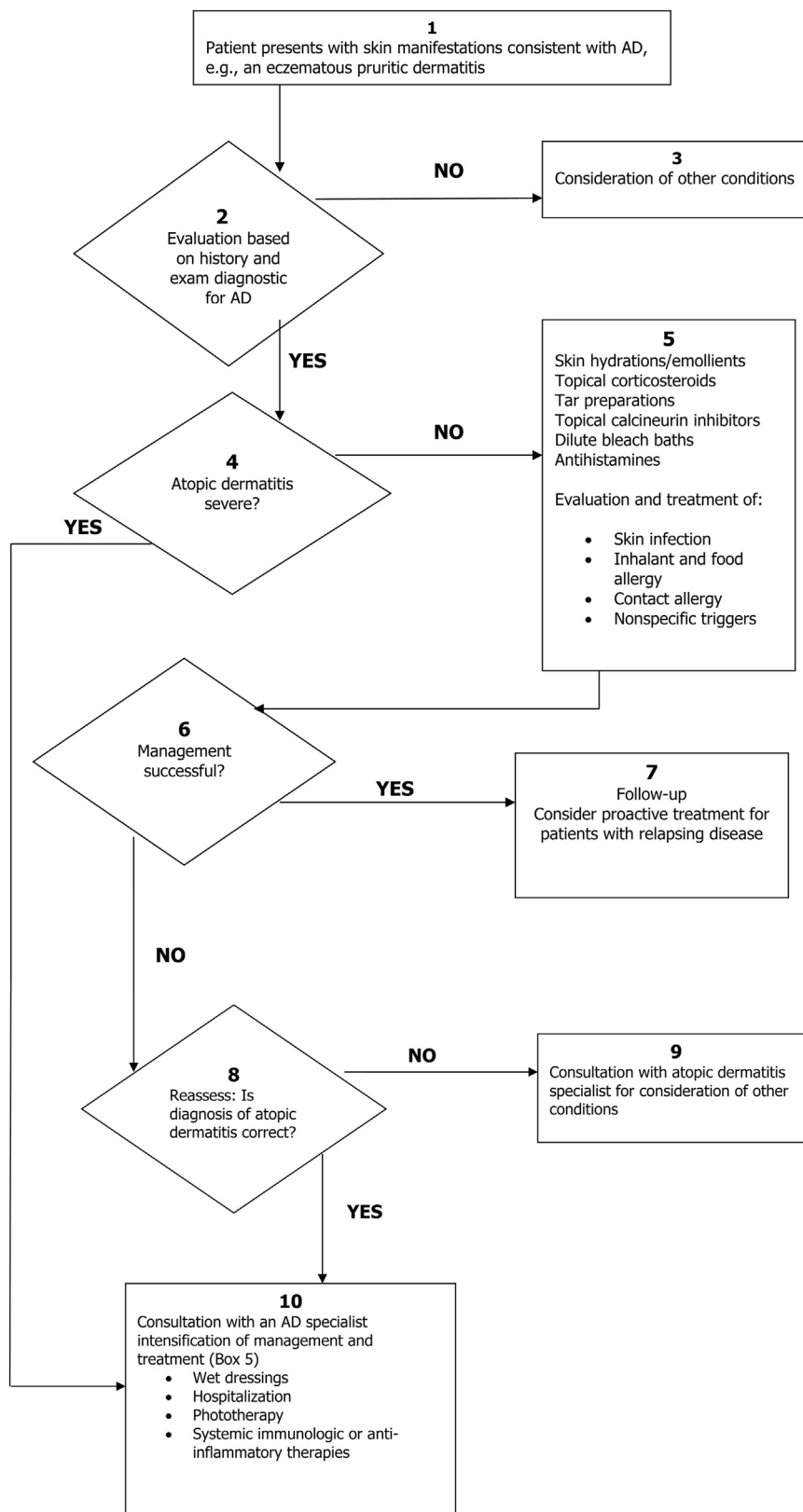


FIGURE 1. Flow chart for the diagnosis and management of AD.

injury.^{10,11} Th2 cytokines then lead to the eosinophilia and elevated IgE characteristic of AD and systemic allergic responses.¹² Chronic AD lesions display a mixed Th1 and Th2 response.¹² Newer data reveal that IL-22 is released in AD skin from Th17 cells and a class of T helper cells called Th22 cells, first identified in patients with AD.^{13,14} IL-22 production stimulated by epidermal Langerhans and dendritic cells results in the epidermal hyperplasia observed in AD and psoriasis, and is intensified in chronic lesions.¹⁴⁻¹⁶

Clinicians are well aware that patients with AD are more vulnerable to infections from staphylococcus, herpes simplex, and molluscipoxviruses. This, in part, is due to innate immune deficiencies in AD skin as well as the reduced epidermal barrier function that results from the Th2 cytokine milieu and primary filaggrin defects.^{17,18} Toll-like receptor activity, important for sensing infection as part of the adaptive immune response, is reduced in AD. Impaired TLR2 and TLR4 expression or activity in AD causes an inadequate response to gram-positive and gram-negative lipopolysaccharides, respectively, and reduced levels of anti-inflammatory IL-10.^{19,20} Recently, mice deficient in TLR4 and Toll-IL-1R domain-containing adapter-inducing IFN- β developed eczematous dermatitis associated with skin barrier dysfunction, cutaneous allergic sensitization, and proinflammatory cytokine production, all hallmarks of AD.²¹ In addition, cutaneous antimicrobial peptides, such as the β -defensins and the cathelicidin LL-37, are inadequate or impaired in AD²² and genetic variations in interferon regulatory factor 2 appear to lead to impaired IFN- γ production against herpes virus infection.²³

Studies of pruritus show that patients with AD experience neuronal sensitization and increased epidermal innervation, so they are particularly prone to itch and discomfort.^{24,25} A recent study found that thymic stromal lymphopoietin not only mediates inflammation in AD, it also mediates cutaneous sensory neuron activation via ORAI1/nuclear factor of activated T cells calcium signaling via ion channel TRPA1-positive sensory neurons.²⁶ Another itch-mediating ion channel, TRPV1, is not stimulated by thymic stromal lymphopoietin but by neuropeptide natriuretic polypeptide b or gastrin-releasing peptide from somatosensory neurons.²⁷ What is fascinating about neuropeptide natriuretic polypeptide b is that it also is produced by cardiac cells to regulate plasma sodium levels and blood pressure; one is tempted to speculate whether this is a molecular connection between emotional stress, itch, and the autonomic nervous system. Interestingly, a study of heart rate variability showed evidence of an excessive sympathetic response to itch and scratching in patients with AD compared with controls with normal skin.²⁸

In summary, the combined effects of skin barrier dysfunction, excessive cytokine-mediated and neurogenic inflammation, reduced antimicrobial function, and chronic scratching promotes a vicious cycle of discomfort and inflammation in AD. It naturally follows that the most effective management of this complicated disease provides a multipronged approach that accounts for patients' molecular and psychosocial milieu.

CLINICAL DIAGNOSIS

Unfortunately, there is no objective test for the diagnosis of AD. Thus, the diagnosis of AD is based on a constellation of clinical features,²⁹⁻³¹ which include pruritus and chronic or relapsing eczematous lesions with typical morphology and

distribution. Acute, subacute, and chronic skin morphologies may be seen, sometimes within the same patient. Acute and subacute lesions are characterized by intensely pruritic, erythematous papules, and plaques that may vesiculate and become exudative. Frequently, excoriations will be found with crusting. Chronic AD is characterized by lichenification (thickened skin with accentuation of the skin markings), papules, and excoriations. The distribution of AD can vary with age as well as with disease activity. In younger children and infants, the scalp, face, neck, and extensor surfaces of the skin are often involved. In older children and adults, flexural areas are more commonly seen with lichenification in addition to acute inflammatory changes.

A method for determination of severity of AD, unfortunately, is not yet universally agreed upon. Many tools exist, including the Scoring Atopic Dermatitis index and the Eczema Area and Severity Index, both of which have been validated and used in many studies.³² Others such as Investigators' Global Assessment, the Patient-Oriented Eczema Measure scale, the Six-Area, Six-Sign Atopic Dermatitis scale, and the Patient-Oriented Scoring Atopic Dermatitis also exist, with varying degrees of validation and usage in studies. Generally, these scales measure the degree and extent of erythema, edema and/or papulation, excoriation, and lichenification, and may variably measure other factors, such as pruritus and sleep loss.^{32,33}

The presence of allergies and allergen-specific IgE also may be a feature and is incorporated into some diagnostic criteria.³⁴ Some investigators have proposed other names for AD without a specific IgE present, such as "atopiform dermatitis" and "intrinsic" AD,^{35,36} but there does not yet appear to be consensus on this point. Filaggrin, although clearly playing a role in a subset of AD, does not have a defined diagnostic role at this time.³⁷ Although there are numerous diseases that can mimic AD, including contact dermatitis, psoriasis, and several syndromes and deficiencies, generally, the diagnosis can be ascertained on clinical grounds.³⁸

Contact dermatitis can be particularly vexing because it can both mimic AD and also may play a role as a trigger and confounder. The confluence of factors that favor the development of contact dermatitis include the impaired barrier function, which may allow increased transmission of allergens, the dysregulation of the immune system already present, the altered bacterial colonization, and the increased exposure to antigens on the skin with the numerous topical therapies applied, including topical corticosteroids themselves.³⁹ Despite these factors, experimental and clinical studies have actually shown a reduced contact sensitivity in individuals with AD.³⁹ Testing for allergic contact dermatitis remains important, however, especially in refractory cases because identification and avoidance of relevant allergens can be critical for success in some cases.

TREATMENT

AD has profound effects on the quality of life of the patient and his or her family, and should be adequately treated to provide continuous relief. In addition, results of a recent study indicate that treatment of AD may actually have positive outcomes on the progression of the disease and the development of related comorbidities, such as skin infections, IgE-mediated allergies, and mental health disorders.⁴⁰ In general, the therapy is dictated by the severity of the disease, the age of the patient, and the distribution and extent of the AD. The concept of a

“therapeutic ladder,” with increasing intensity of management for more severe or refractory disease also may be useful.⁴¹ At a minimum, the ADPP multipronged approach that includes skin hydration, topical anti-inflammatory medications, antipruritic therapy, antibacterial measures, and the elimination of exacerbating factors addresses numerous components of the disease and appears to have powerful synergistic effects (Figure 1).

Such a concerted plan can be very complex, and a written “eczema action plan” akin to an asthma action plan may be useful in consolidating the therapies and improving understanding and adherence (Tables I and II).^{42,43} Encouraging families to call with questions or concerns is paramount to their adherence to the plan. Reviewing the written eczema action plan with the family by telephone often clears up any miscommunication or misunderstanding. It is important to review the eczema action plan at follow-up visits, especially if changes have been made.

Skin hydration

AD is characterized by impaired skin barrier function, with resultant xerosis. Moisturizer application, particularly after soaking in water for 20 minutes can help retain water and improve barrier function.^{44,45} Importantly, there are measurable clinical ramifications to moisturizer use, including powerful itch relief,⁴⁶ a corticosteroid-sparing effect,^{47,48} and significant improvement of disease when used regularly.⁴⁹ Although there are a wide array of different moisturizer formulations, including prescription-only “barrier creams,” there are very little comparative data that any are significantly better than others.⁵⁰ Until there is evidence that favors a particular type of moisturizer, allowing patients to find moisturizers that they find soothing and that they will actually use may be the most practical approach. If professional samples of various moisturizers are available in the office, then encourage the patient to try them while in the office. This gives the provider opportunities for important educational points, because the patients may not be applying moisturizers effectively.

Children may resist baths and application of moisturizer due to stinging on application or finding emollients messy or greasy.^{51,52} Taking a bath with clothes or pajamas on will often work as a distraction and provide a placebo effect, especially if the child complains of stinging. Strategies such as games, distraction, and involving children in skin care may reduce anticipatory anxiety and increase cooperation.^{52,53} For younger children, it may help to have the child assist in applying moisturizer, apply moisturizer to the parent first, or “draw” on the skin by using the moisturizer. Use of bath toys and developmentally appropriate explanations of the rationale for baths (eg, “a drink for thirsty skin”) may also be helpful. For older children and adolescents, use of a lighter moisturizer for the face or during the day may increase adherence.

Topical corticosteroids

For mild AD, simply using moisturizers may be enough to calm the skin and prevent flares. However, should moisturizers alone not suffice, then topical corticosteroids are the mainstay of effective treatment for AD.⁵⁴ These can be applied to the eczematous areas once or twice daily when needed to calm both itch and inflammation, and to allow skin healing in addition to continued moisturization. Although powerful, corticosteroids must also be used with caution: adverse effects are directly related to the potency and the duration of use. Judicious use of higher potency

TABLE I. Key points for care of AD

Skin care plan should include instructions for
Bathing daily
Skin cleansing
Skin hydration, schedule for emollient usage
Schedule for appropriate use of topical anti-inflammatory agents
Plan for control of itching and infection
Wet wrap use if prescribed
When to contact provider
Patient education tips
Simplify regimen
Consistent schedule for skin care and bedtime
Frequent moisturizing
Importance of follow-up

TABLE II. Revision of Table I: a sample eczema action plan

When severe (itchy, red, oozing)
AM
1. Apply topical medication to eczema areas
2. Apply moisturizer liberally
3. Take vitamin D supplement
During the day
1. Apply moisturizer liberally
PM
1. Soak in dilute bleach bath for 10 min
2. Pat dry but while still damp do step 3
3. Apply topical medication to eczema areas
4. Apply moisturizer liberally
*Do this for several days (up to 1 week) until better
When better
AM
1. Apply moisturizer liberally
2. Take vitamin D supplement
During the day
1. Apply moisturizer liberally
PM
1. Soak in dilute bleach bath for 10 min twice weekly
2. Pat dry but while still damp do step 3
3. Apply moisturizer liberally

*Do this for several days (up to 1 week) until better.

preparations in terms of avoiding thin-skin areas such as the face or skin folds, and minimizing the duration (ideally 1-2 weeks maximum at any time) is critical. Once improved, there is evidence that supports use of a lower-potency corticosteroid, for example, fluticasone, twice weekly to eczema-prone areas. This so-called proactive therapy has been shown to decrease flares, improve quality of life, and lower cost and amount of medication exposure in more severe cases.⁵⁵ Patient and parent concerns about adverse effects and confusion regarding differing potencies are common barriers to the use of topical corticosteroids as prescribed, which suggests the need for adequate education on the safety and appropriate use of these medications.^{56,57}

Topical calcineurin inhibitors

There are situations when topical corticosteroids may not be appropriate or must be discontinued due to risk or adverse effect.

Fortunately, the topical calcineurin inhibitors represent another class of anti-inflammatory medications that can be used similarly but without the same adverse effect profile. Tacrolimus and pimecrolimus inhibit the activation of key cells involved in AD, for example, T cells, dendritic cells, and mast cells.⁵⁸ There is abundant clinical data that these medications are safe and effective in treating AD in patients 2 years and older.^{51,59} A local burning sensation, particularly when applied to broken skin and often only for the first several days of use, is the only common adverse event. Unlike topical corticosteroids, these medications do not cause skin atrophy and thus may be favored for the treatment of facial and eyelid eczema. In clinical trials, tacrolimus appears to have an anti-inflammatory potential similar to a midpotency corticosteroid, with pimecrolimus being slightly less potent.^{60,61} However, certain patients may respond more favorably to a topical calcineurin inhibitor.⁶² Proactive therapy also has been defined for these medications: twice weekly application to eczema-prone areas has been shown to reduce flares, similar to the fluticasone data presented above.⁶³

Despite the black box warning on this class of medications since 2006, a nested case-control study of almost 300,000 patients with AD found no increase in risk of lymphoma in patients treated with topical calcineurin inhibitors.⁶⁴ Reviewing with the family that the topical calcineurin inhibitors will be used with careful monitoring and follow-up is helpful. Reviewing the black box warning and the recent reassuring research will aid adherence.

Antihistamines

Although frequently prescribed for patients with AD, a review of 16 controlled studies found little evidence for the effectiveness of oral antihistamines on the itch of AD.⁶⁵ This is perhaps not surprising because histamine is only one of the mediators that can induce pruritus during an inflammatory response. However, there are other aspects of antihistamines that may justify their use in AD. For patients with concomitant urticaria or allergic rhinitis, there may be benefit in controlling possible triggering events,⁶⁶ and, because the pruritus is often worse at night, a sedating antihistamine may allow for improved rest.

Vitamin D

For a relatively benign intervention, there remains some controversy about vitamin D supplementation in AD. The observation that AD is often worse during the winter months led to a trial that showed significant improvement in children supplemented with vitamin D (80% with improvement vs 17% of the control group).⁶⁷ This has been corroborated by studies that demonstrate an inverse relationship between vitamin D level and eczema severity.⁶⁸ The possibility for benefit seems to greatly outweigh the negligible risks of vitamin D supplementation, especially in those patients who report worsening in the winter and in any patient with known deficiency.⁶⁹ Conversely, topical vitamin D preparations are to be avoided because there are reports that they worsen eczematous dermatitis via both allergic and irritant mechanisms.^{70,71}

Treatment of infections

Staphylococcus aureus is known to play a significant role in AD, frequently causing infections but also driving inflammation, even as a colonizer.⁷² Nonetheless, results of a large review failed to find evidence that common antistaphylococcal treatments were helpful in uninfected eczema.⁷³ Staphylococcal infections may be

severe and recurrent. When frank infection is present, a short course of an appropriate oral antibiotic is warranted. In areas where there is a high prevalence of methicillin resistance, taking a skin culture before empiric treatment may help guide therapy selection. Encouraging the family to call when there is no significant improvement or there is worsening of the skin infection will help to ensure adequate treatment.

Dilute bleach (sodium hypochlorite) baths may reduce the need for systemic antibiotics and have been shown to significantly decrease AD severity.^{74,75} As mild as swimming in a chlorinated pool, this gentle and inexpensive therapy can probably be traced back to World War I and the use of Dakin solution for infected wounds. Mixing one-fourth to one-half cup of plain household bleach into a 40-gallon bath and soaking twice weekly or more can result in dramatic clinical improvement in some patients. Explicitly describing the similarity of bleach baths to use of chlorine in swimming pools may help reduce patient and parent concerns and increase use of this therapy.

Wet dressings

Best reserved for patients with difficult-to-manage disease, wet dressings ("wet wraps") in combination with topical corticosteroids represent an extremely powerful therapy. This technique generally entails soaking the skin, applying a topical corticosteroid to the affected areas, then applying a damp layer of gauze or clothing, followed by a dry layer. Wet dressings help with skin barrier recovery, increase the efficacy of topical corticosteroids, and simultaneously protect the skin from scratching, which may allow for more rapid healing.⁷⁶ Wet dressings appear to be safe for up to 14 days and actually have been shown to decrease the amount of corticosteroid used.⁷⁷ However, wet dressings should not be overused because they can result in skin maceration, secondary infections, and, rarely, systemic corticosteroid adverse effects. A detailed description of wet-wrap therapy may be found in Boguniewicz et al.⁵³

Acceptance of wet dressings by children may be increased by strategies such as "wrapping" a doll⁵³ or introducing wet dressings as "mermaid pajamas" or a "superhero suit." Explaining to parents the concept of "cooling" the skin and providing another barrier or layer to discourage scratching helps to bring the parents onboard. Parents also should be prepared that it is normal for children to take several days to get used to wet dressings and that the wet dressings should not be discontinued prematurely. In addition, it is important to explain to families that the child will not develop "pneumonia or an upper respiratory infection" from going to bed with damp clothing. By taking the dry clothing directly from a warm dryer provides a "sauna effect" and can help the parents to feel better about applying damp clothing to their child.

Trigger elimination

A large part of the frustration with AD is the fact that there can be numerous triggering factors. These range from common irritants, such as soaps, chemicals, and certain fabrics,⁷⁸ to more specific food and environmental allergens.⁷⁹ There is evidence that some proteins, such as those produced by house dust mites, have innate proteolytic activity that can damage the skin barrier, even in the absence of actual allergy.⁷² Despite this, interventions with house dust mites avoidance have not demonstrated a strong effect.⁸⁰

Food triggers for AD are often suspected, and nearly one-third of patients with moderate-to-severe AD have verifiable type I hypersensitivity food allergies that could trigger a flare.⁸¹ However, there is some evidence that concerns about food may be magnified compared with actual risk.⁸² Many trials examined dietary exclusions to improve AD in adults and children. There is evidence to support improvement in those who were avoiding foods to which they have positive IgE status but no benefit in elimination diets in those without known IgE-mediated food allergies.⁸³ Testing for food allergy is generally recommended only for children younger than 5 years old who have had persistent AD despite optimized therapy or a reliable history of immediate allergic reaction to a food.⁸⁴ Reviewing with the family the difference between an IgE-mediated food reaction and an irritant reaction is often helpful in allaying the family's fears. Applying a petroleum base to the child's face and hands before eating creates a barrier that may help to decrease irritant reactions.

For patients with severe and refractory disease, after attempting to maximize the above modalities, occasionally more aggressive systemic treatments are required. Phototherapy and systemic immunomodulating agents, such as methotrexate, cyclosporine, mycophenolate, and azathioprine, may be necessary, although these agents must be used with caution and by an experienced clinician because the potential for adverse effects is considerable. For a fuller discussion of each agent, we would refer the reader to the practice parameters, which has a section devoted to systemic immunomodulating agents and phototherapy.¹

IMPROVING QUALITY OF LIFE

The impact of AD on quality of life is well documented, with children and parents reporting itching and scratching, and associated sleep disruption to be among the most problematic aspects of the condition.^{85,86}

Sleep

Sleep disruption caused by pruritus and scratching is common among children with AD and includes difficulty falling asleep, frequent awakenings, overall reduced sleep efficiency, difficulty waking in the morning, and daytime tiredness.⁸⁷⁻⁸⁹ Awakenings may persist even in remission, which suggests the role of learned sleep patterns in maintaining sleep disturbances for some patients.⁹⁰ Sleep disturbances also have been associated with increased daytime behavior and mood problems, and may mediate an increased risk for attention-deficit/hyperactivity disorder in children with AD.^{89,91}

Sleep often improves with effective anti-inflammatory treatment of AD,^{92,93} and use of wet wrap therapy at bedtime might be helpful in reducing pruritus and by serving as a barrier against scratching.⁵³ As outlined in Table III, sleep hygiene should be optimized for all patients (eg, maintenance of a consistent sleep schedule for bedtime and naps, a relaxing bedtime routine).⁹⁴ If sleep problems persist after the skin is under good control, then it may be helpful for families to consult with a psychologist or sleep specialist, given strong empirical support for behavioral treatment approaches to bedtime problems and night awakenings.⁹⁵

There are limited data regarding the safety and effectiveness of pharmacologic treatment of sleep problems for patients with AD and children in the general population.^{94,96} Sedating antihistamines might offer an advantage for some patients when used at bedtime, and other therapeutic agents might be useful on a

TABLE III. Practical strategies for managing itch and sleep disruption

Sleep disruption	
1. Optimization of sleep hygiene	
a. Consistent schedule for sleeps and naps	
b. Relaxing, consistent bedtime routine	
c. Avoidance of screen time and/or electronics before bed	
d. Attention to sleep associations after skin improved (phasing out the need for parental presence at bedtime)	
2. Use of relaxation strategies for sleep onset and focusing of attention away from itch (downloadable recordings and CDs available for adults and children)	
3. Covering of skin to reduce skin damage due to nighttime scratching	
a. Wet dressings as necessary	
b. Cotton pajamas with long pants and sleeves	
c. Eczema "sleepsuits" available for purchase online	
d. Coverings for hands and feet (or modified pajamas with socks or gloves sewn or taped to pajamas)	
4. Consideration of sedating antihistamines	
5. Consultation with sleep specialist or psychologist for persistent sleep problems	
Itching and scratching	
1. Nonblaming approach to itch (focus on what patients can do when itchy)	
2. Use of elements of skin care plan (baths, moisturizer, medication, wraps)	
3. Covering of skin (long clothing, gloves, tights, wraps)	
4. Distraction and redirection to hands-on activities	
a. Hand-held electronics, blocks, crayons, stress balls, knitting	
b. Special toys for baths (bath crayons)	
5. Use of competing sensory sensations (cool pack, cool washcloth)	
6. Use of psychological interventions to reduce stress and focus attention away from itch	
a. Relaxation strategies (guided imagery, relaxed breathing, muscle relaxation, biofeedback)	
b. Cognitive behavioral therapy to address stress and anxiety	

short-term basis,⁹⁶ particularly when used in conjunction with nonpharmacologic approaches (eg, good sleep hygiene, parent and/or patient guidance, behavioral treatment).⁹⁴

Itching and scratching

Breaking the itch-scratch cycle is an important component to management of AD (see Table III for practical tips). Because stress has been shown to be a trigger for AD, psychological interventions, such as relaxation therapy, stress management, behavior displacement, and cognitive-behavioral therapy, may be effective in reducing itch and scratching behavior associated with AD. A referral to a psychologist may be considered for patients who have difficulty with emotional triggers of scratching. There also are downloadable relaxation resources for children and adults that are easily accessible and may be useful for some patients. Parents may benefit from guidance in strategies to reduce children's scratching behavior, such as distraction and redirection to hands-on activities.^{97,98} Some patients find competing sensory sensations soothing when itchy, such as use of a cool washcloth or cool pack to the itchy area. In general, a focus on what patients can do when they are feeling itchy is likely to be more effective than a focus on the scratching behavior itself.

Psychological impact

Patients with AD may be at a higher risk for anxiety, depression, and attention-deficit/hyperactivity disorder,⁹⁹⁻¹⁰² and may experience embarrassment about the condition of their skin during flares.⁵⁷ Psychological evaluation and/or therapy may be considered for patients who are experiencing psychological distress associated with AD or for whom mood or anxiety influences the course of the disease or adherence to recommended treatments.

SUMMARY

Fortunately for the vast majority of patients, a combination of the above therapies allows for excellent disease control to be obtained. Frequent follow-up appointments and ongoing education help the family to manage flares and maintain skin integrity. These treatments, along with education and support, can make a tremendous difference in the quality of life of patients with eczema. Future therapies will aim to target some of the molecular pathways described, but patients will likely always need a multifaceted plan that combines medical, psychological, and social interventions.

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